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ABSTRACT

The literature on teaching reading to brain-damaged children was reviewed for the period 1960 to 1970. Only nine articles represented experimental investigations of the problem. These articles were examined with respect to the adequacy of reporting information and data concerning the diagnosis of brain damage. The criteria for diagnosing brain damage were generally inadequate or nonexistent. There was little evidence to suggest that children with chronic neurological impairment at the level of the cerebral hemispheres require or benefit from teaching procedures which differ from those used for reading retardates without brain damage. Advantages and limitations of various neurological tests are discussed. Recommendations are made for standards to be followed in documenting brain damage in research studies on the teaching of reading to brain-damaged children. A bibliography is included. (Author/CM)

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Teaching Reading to Brain-Damaged Children: A Review

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The evidence that retarded readers with brain damage require special methods of instruction is extremely meager. The lack of evidence stems from a paucity of studies which directly address the question of differential instruction as a function of brain damage. Studies that have attempted to investigate this problem suffer from an inadequate experimental design and a failure to specify the criterion information used in the diagnosis of brain damage.

In this paper we have reviewed the experimental studies on brain damage and reading which have appeared in the literature between January 1960 and July of 1969. Our purposes were to examine the evidence for not only (a) the effectiveness of various instructional methods for teaching brain injured children to read, but also,

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and primarily (b) to assess the quality of the criteria used in the documentation of brain damage, and then (c) to make recommendations concerning standards to be employed in future investigations of remedial techniques for the brain-damaged.

Does the fact of brain damage itself make a difference? If one views the number of instructional programs and special classes for the perceptionally handicapped, the hyperactive child, the child with minimal cerebral dysfunction, etc., the answer can only be given in the affirmative. The scientific evidence is lacking. As Birch (1964) has pointed out, the "brain-damaged" child may or may not have damage of the brain. Cruickshank (1966) stated that there is no agreement on the meaning of the term. Others have implied that the characteristics of "these children" are well known and that it is immaterial whether or not there is damage to the brain. There are flaws in this line of reasoning. First, such a statement shows little respect for the value of scientific investigation and the rules of evidence which permit advances in knowledge.

Second, if one assumes that it is immaterial whether or not there is damage to the brain, one has indeed made a diagnosis, namely, that damage, if present, is chronic and non-progressive.

Knowledge of neurological deficits may or may not help in planning an educational program. One will never know unless rigorous experimentation is done, and such experimentation must include a clear delineation of the criterion information by which brain damage is specified. Possibly children with lesions primarily involving the left cerebral hemisphere would indeed profit more from a teaching regime designed to promote language skills than would children with lesions primarily involving the right cerebral hemisphere. The validity of this statement can never be judged unless formal experimentation is done in accordance with commonly accepted criteria of good experimental design.

In order to provide a background for the review of the studies pertaining to brain damage and reading, it will be necessary to

consider (a) some of the problems in identifying the brain-damaged child, (b) the procedures involved in a neurological examination, and (c) the limitations of various neurological criteria intormation. In the section which follows, we have not discussed the use of psychological tests for inferring cerebral dysfunction. If one uses psychological tests to infer neurological deficit, the criterion becomes the psychological tests and there are the attendant problems not only of the reliability but also of the validity of the classification. Psychological tests can be used to describe the ability deficits, the emotional characteristics or the perceptual handicaps of children with brain damage, but to do so, there must be evidence independent of psychological tests for the fact of damage, otherwise, the prophecy becomes self-fulfilling. It is, for the purpose of this discussion, meaningless to ask whether psychological tests are better than neurological tests for identifying brain damage, because if the criterion is neurological information, the procedure cannot be better than the criterion.

Limitations of Neurological Evidence

Problems in Specifying Brain Damage

A major difficulty in studying the relation between brain damage and reading abnormality is one of definition. The term "brain damage" by strict definition implies a "loss due to injury". Loss may imply loss of brain (neurones, glia, and blood vessels) and if sufficient brain cells are lost, there is an associated loss of specific function, or deterioration in the quality of an existent function. Neuronal loss is irreplacable and the associating functional deficit would therefore be permanent. On the other hand, "loss due to injury" may imply another thing, i.e. loss of function. Thus, a patient with a post infectious meningoencephalitis may have diffuse brain pathology involving the meninges, the subpial and subependymal, and peri- and paravascular gray and white matter. This pathology may be accompanied by loss of consciousness and a diffusely slow electroencephalogram (EEG). Within weeks, consciousness can return, the EEG frequencies become normal and the patient can

function as before. This sequence represents a transient loss of function by a large part of the brain due to reversal of pathology. There need not, in such instances, be permanent evidence of functional or structural loss. In summary, brain damage may imply cellular loss and an anatomical change which, if large enough, will produce clinical symptoms. Depending upon the anatomical site of the structural change there may be focal or diffuse signs of malfunction which are permanent. On the other hand, brain damage may imply functional loss with an anatomical change which is reversible and thus a loss of function which is similarly reversible.

The basis of the discussion so far is the assumption that specific symptoms and signs are consistently associated with pathological change and malfunction at the cellular or ionic level of a particular level of the brain. This is so to a usable degree when one is involved with "classical" neurology. This, if a 10-year-old child suddenly has convulsions involving the right face, arm, and leg, and following recovery from the seizure has a permanent right hemiparesis, mild

dysarthria, and persistent total awareness with no evidence of brain stem dysfunction, one knows that the patient has a lesion involving the left precentral gyrus and posterior frontal lobe or the white matter tracts eminating from these areas and going through the left internal capsule. Further investigation must be undertaken in order to determine the nature and extent of the lesion and whether or not it is due to focal inflammatory disease (abscess), vascular disease (occlusion of a branch of the middle cerebral artery by vasculitis, emboli, or a rupture of a vascular abnormality), tumor growing within the brain substance (glioma) or upon the surface of the brain(meningioma, chronic subdural effusion). The delineation of the lesion causing the signs of pathology will necessitate the use of a number of ancillary techniques including a lumbar puncture, electroencephalography, echoencephalography, contrast studies of the brain vascular system and/or brain ventricles and radioisotope encephalography (Toole, 1969). Despite the use of all of these and the delineation specifically of abnormality of electrical discharge

from the involved area, abnormal permeability of the vascular system in a focal area by radioisotope distribution, and abnormalities of displacement of the midline structures by echoencephalography, displacement of vessels by arteriography or of the ventricles by air contrast studies the precise nature of the lesion may have yet to be determined by surgical exploration and microscopic examination of removed tissue.

Unfortunately, signs of neurological dysfunction associated with reading problems in children are either absent or when present are not examples of classical neurology. Instead, the signs are those of mild brain dysfunction. These signs are so difficult to classify that they have been the subject of several national task forces which have undertaken to define and describe their existence under the heading of the syndrome of Minimal Brain Dysfunction (Clements, 1966, Task Force II Report, 1969). As presently understood the syndrome consists of children with near average, average, or above average intelligence who present

with learning and/or behavior disabilities associated with deviations of function of the central nervous system. These deviations are manifested by various combinations of impairment of perception, conceptualization, memory, language, motor coordination, and control of attention and impulse. The neurological signs of this syndrome[1] are highly variable and include some combination of the following; abnormalities of eye movement, head-eye dissociation, articulation, alternating supination and pronation of the extended arms and hands, serial apposition of fingers, heel-shin tapping, walking on heels and toes, hopping on one foot, and tandem walking. In addition, short attention span, easy distractability, and difficulties with visual-motor tasks can be found. These disabilities have several qualities; first they are often classifiable as disabilities only when compared with a rough age dependent standard, i.e. the 7-year-old may perform like a 4 or 5-year-old; second, as the child grows older, abilities to perform tests of integration of movement improve; third, there is no known brain pathology asso-

[1]These signs are the so-called "soft signs", a term we find objectionable.

ciated with these aberrations and none can be implied by correlation with knowledge of "classical" neurology; and finally, some children have behavior or learning disabilities without these signs and some children with poor performance in the motor tests have no clear learning or behavior abnormalities.

Although the syndrome has been carefully defined as that of "minimal brain dysfunction" it is an habitual tendency to regard these children has having brain damage despite the lack of evidence for structural damage. This is done we believe because of an uncritical tendency to equate poor psycho-motor function with a damaged brain. Another reason why this group of children has been labeled "brain damaged" stems from an early and widely accepted concept of Strauss and Lehtinen (1947) that children who behaved in a specific fashion and have a history of antecedent disease which could have produced brain damage had a syndrome of behavior due to "brain damage". At the same time, similarly behaved children who do not have a history of antecedent disease which could have produced brain damage

but behaved in a similar manner have also been labeled as children having the "brain damage syndrome". The problem boils down to the fact that children with minimal brain dysfunction syndrome have evidence by examination of abnormal neurophysiology, principally in tests of motor integration, impulse inhibition, maintenance of attention, and learning abilities. Whether the basis of this abnormal function lies in a delay of brain maturation, in an as yet undescribed tissue or chemical pathology, or some other factor, is not presently known.

Assuming that we cannot consistently find cases of classical neurological syndromes associated with the common types of reading problems in children, what kind of information should we seek in the study of children with reading problems which might define the potential for, or existence of, concomitant brain damage or neurological dysfunction? What is the value of each piece of information and what is its significance?

Specific Neurological Tests

The most frequently used pieces of evidence to determine the existence and possible cause of brain dysfunction are patient history, physical and neurological examination, electroencephalography, and neuropsychological tests. Contrast studies of brain vasculature and brain ventricles (arteriography, pneumo- or ventriculoencephalography) or isotope encephalography can show very specific evidence of abnormalities of structure, and by inference, brain damage, but these studies are used principally in the instance when the nature of the disease may be one which should be treated to prevent further damage or when repair of existing pathology is possible. Since this situation rarely exists in the type of patients under discussion these tests are rarely used. Since the use of neuropsychological tests is discussed elsewhere, we will now turn our attention to the value of history, neurological examination, and electroencephalogram in the investigation of patients with a reading problem.

Historical data providing evidence of possible value in relating reading problems to evidence of brain dysfunction include items in the family, gestational, paranatal, developmental, and other past history. In using these data, one must be aware of the limitations. For example, a history of a well documented incidence of developmental dyslexia in a male uncle who went through college but with great effort and using as many auditory input crutches as possible (tape recorder, discussion with classmates) is one thing, but a history of an aunt who read poorly and dropped out of 7th grade at age 18 years, is another. A history of vaginal bleeding intermittently during the first trimester of pregnancy or of a full-term, 4-day-old infant with a serum bilirubin of 18 mgs.% which was untreated are both pieces of evidence which increase the likelihood of the infant in one instance having congenital encephalopathy at birth and in the other, developing bilirubin encephalopathy. However, neither guarantees the cause and effect relationship between the event and the finding later in life of reading difficulty or signs of minimal brain

dysfunction. A history of delayed onset of respiration at birth, of treated bacterial meningitis at age 3 years, or of brief unconsciousness following a fall from a table at 20 months of age are all examples of statistics, which if coupled with another observation, may have possible relevance to the subject at hand. Minimal brain syndrome or abnormalities of reading may be related to such antecedent events, but when the past history lists items that have a statistical possibility but not an absolute chance of causing MBD or reading problems, the limitation of information must be stated.

The ability of one to deduce from the neurological examination evidence of structural brain damage or of neurological dysfunction without evidence of clear structural damage has been previously described. Several points need emphasis, however. First, since a classical neurological syndrome which implies damage to a particular area of the brain will be an uncommon finding in children with reading problems and since evidence of neurological dysfunction of varying severity and region of involvement will be common, it would

be best in reporting these data to describe the tests used and to grade the level of dysfunction. The second point is that since the signs and symptoms of dysfunction change in severity with age, these data should be grouped according to age and the change in severity of the signs and symptoms from one time period to another for each individual should be documented.

The ability of the EEG to determine the existence of brain damage or neurological dysfunction is limited. Some of these limitations are unique to this test and some are similar to those of the neurological examination. It is rare that the brain wave test could be used alone to determine the existence of brain damage in a structural sense. If one assumes a certain wave form is an abnormal one and that this type of electrical discharge indicates disordered electrophysiology, hence functional damage, a further point to be determined is the significance of this finding for the patient. To put these problems in perspective, let us review the subject briefly.

The electroencephalograph is an electrical amplifier attached to an oscilloscope which measures the frequency and voltage of oscillating potentials derived from pairs of electrodes placed on the surface of the scalp. The electrical potential originates from the electrical activity of the brain beneath. The normal frequencies and voltages vary with the age of the patient, the stage of consciousness, and the area of the brain being measured. Electrodes placed over the surface of the scalp cover only 1/3 of the total brain surface and it is unclear how much of the total brain electrical activity is measured because of the spatial limitation. Furthermore, it is unclear how much of the electrical activity of the brain at great distances from the surface electrode is measured at the scalp. This situation probably accounts in part for the problem posed by patients who have gross pathology such as intracerebral hemorrhage, deeply situated tumors, or discharging electrical focus in the amygdala and a normal EEG (Glaser, 1963).

Interpretation of the EEG is difficult. Standards of normality vary according to the patient's age and state of consciousness. The

detection of abnormalities may depend upon the recording technique for certain abnormalities may be recognized only in monopolar not bipolar tracings, and in drowsiness and sleep and not when the patient is awake. Some wave forms are seen much more frequently in sleep than wakefulness. Because of the large number of variables to be considered in the interpretation of the EEG, the readings may be biased according to the experience and emphasis of the interpreter.

The significance of abnormalities in the EEG which are clearly established is not always easy to determine even in the light of clinical data. For example, a patient with grand mal epilepsy or brain tumor may have a normal EEG. On the other hand, a patient with 14 & 6/second positive spikes may have no symptoms, severe behavior problem, recurrent attacks of headache, vomiting and sleep, or a blatant seizure disorder (Gibbs & Gibbs, 1964). A negative spike discharge occurring in the EEG, especially when repeated, and from one locus, is thought to indicate an abnormally discharging

area of the cortex. Such a negative spike focus can occur in a child who has suffered brain trauma in the past with resultant focal cortical damage. Historical and clinical evidence of brain injury is thus available. Under such circumstances, the focal discharge is presumed to derive from the area between the scar tissue, replacing damaged brain, and the surrounding normal brain. However, similar spike foci in the EEG can be seen in children who have seizure disorders and no history of antecedent disease leading to brain damage or concomitant neurological abnormality on examination. Furthermore, repeated EEG tracings over a period of years reveals in some of these children that the spike focus moves from one area of the brain to another and finally disappears altogether (Gibbs, Giller & Gibbs, 1954). The occurrence of shifting spike foci in children with seizure disorders is thus evidence that the cortically evoked abnormal discharge is not a sign of structural brain damage but rather evidence of abnormal cortical electrophysiology, the molecular basis of which is not yet known.

The difficulties encountered in relating EEG abnormalities to a dysfunction which includes reading problems are illustrated by one study of electroencephalography and learning disabilities (Hughes, 1968). Among 66 children who were studied, 62% had abnormal EEGs. The most frequent EEG abnormality was posterior slowing and 14 & 6/second positive spikes. An attempt was made to correlate the EEG findings with 22 psychological test functions. Relations between various combinations of EEG abnormality and psychological test abnormality were discovered only after grouping and regrouping the two sets of data. It was concluded that relationships between EEG and psychological function in a population of learning disorders could be obtained with multivariate techniques using the aid of a digital computer and that more than univariate analysis was necessary to obtain significant results. However, the significance of these results is quite puzzling and at present merely raises more questions than it answers. The complexity of translating this type of data into practical formulation is enormous.

Such studies are clearly dependent, whatever one may think of their implications, upon precisely defined terms and there is no room for issue clouding ones such as "brain damage" to explain the results.

In conclusion, the EEG serves best in studies of children with reading disabilities if it is regarded as an instrument and can reflect a sign of disordered electrophysiology. The EEG sign must be clearly defined, its relation to other parameters must be described and cognizance taken of the fact that this sign can change with time and is not in itself an indication of structural brain damage. In itself, the EEG can neither diagnose MBD syndrome, reading problems, learning disorders, or brain damage.

The contribution of the neurologist and his techniques of history taking, patient examination, and the use of the electroencephalogram to studying the relation between brain damage and reading disability can be a significant one. It is based mainly upon insistence that the methodology, implications and limitations of each of the techniques used be clearly known and described. It insists upon

the recognition of the implications of the term "maturation" in studying the population which is involved, namely children. Constant insistence upon the need to apply these principles will certainly increase the volume of reliable conclusions derived from future studies attempting to relate reading problems to real brain damage or the more common neurological dysfunction.

Remedial Reading Procedures for the Brain Damaged

With the foregoing knowledge of what constitutes adequate neurological information, let us now turn to an examination of some of the specific studies which pertain to the question of brain damage and reading.

In selecting articles for this presentation, we used the standard bibliographical references--The Psychological Abstracts, the Readers Guide to Periodical Literature, and the Annual Summaries of Reading Investigation by Helen Robinson et. al. If the title of the article contained terms such as "dyslexia", "perceptually handicapped child", "brain injured child", "minimal cerebral dysfunction"

or one of the multitudinous synonyms, the article was included for review. In all, 106 articles were chosen. Thirty-six of these were position or discussion papers on dyslexia, learning disabilities and brain damage. There were 23 articles which were descriptive studies of dyslexic and/or brain-damaged children. That is, the studies were concerned with the psychological characteristics or the ability deficits of children who had been diagnosed as dyslexic or brain-damaged. The actual number of articles appearing in the foregoing categories during the 1960s was much larger but we eliminated many from consideration because they were primarily tangential to the purposes of this paper. Forty-two articles pertained to educational and remedial methods for the brain-damaged child and of these 42, 33 described teaching procedures but gave no evidence of their merit. Only nine, (representing seven studies) were honest efforts at experimental investigation of the efficacy of a particular method or methods of instruction.

Research Articles

These seven studies were selected for review and our criticisms have been limited to descriptions of the sample employed and the quality of the neurological criterion information. Some may view this as a rather restricted task. Many of the studies described could also be criticized and, perhaps more severely, for inadequate statistical analysis, the lack of an appropriate control group, or the failure to employ randomization. We selected the aspect of the neurological criterion information as our focus because many researchers in reading lack training in the neurological sciences and are unaware of the merits and limitations of diagnostic neurological information. They do not know what constitutes acceptable evidence to neurologists. In addition, even if the study were otherwise adequately designed and analyzed, failure to provide satisfactory criterion information would restrict the generalizations which could be drawn. Indeed, it is of questionable benefit to know that teaching method A is more effective for Group Y, while teaching

method B is more effective for Group Z, if one could not describe how Y and Z differ. Finally, if one accepts the truism that the purpose of scientific experimentation in education is to generalize to the "not here" and the "not now", there is a need to scrutinize the factual basis for such generalizations by critically examining, from a neurological point of view, the investigations on brain damage and reading where it is highly questionable as to just who was studied.

A major study and one of the more conscientious attempts to investigate specific teaching procedures was reported by Cruickshank, Bentzen, Ratezeburg and Tannhauser (1961). They listed the psychological characteristics of the brain-injured child as distractibility, motor dysinhibition, dissociation, disturbance of figure ground, perseveration, and poor self concept and body image. Their sample consisted of forty subjects divided into two diagnostic groups, (a) those clinically diagnosed as having neurological and medical evidence of brain damage and (b) those with behavior patterns

typical of the brain-damaged group, but no evidence of brain damage on medical examination or history. For each subject, the authors list the results of the clinical neurological examination. They also present perinatal data. Unfortunately, the authors do not indicate the specific criteria for brain dysfunction but they do state which of the subjects were diagnosed as having neurological deficits, and the careful reader, by scrutinizing the results of the clinical neurological examination, can form an independent opinion as to whether the information was adequate. In the experiment, the authors had two experimental groups and two control groups, each experimental and each control group contained an equal number of brain-damaged and non-brain-damaged subjects. The authors did not report the results according to brain damage vs. non-brain damage so there is no way to judge the efficacy of their procedures for brain-damaged children. However, the authors are to be complimented on the care with which they presented neurological criterion information. One may not agree with the adequacy of the information, but the data are sufficient so that one has a basis for making a disagreement.

Talmadge, Davids and Laufer (1963) studied experimental methods for teaching emotionally disturbed, brain-damaged retarded readers. They studied a group with reading impairment due to central nervous system dysfunction, subcortical or cortical, which has interfered with the ability to retain and reproduce visual, auditory, or motor cues as well as inducing behavior syndromes of hyperactivity, distractibility, perseveration, and a lack of abstraction and generalization. From 42 children, they selected 24 who were found to be at least two years retarded in reading on the California Achievement Test. These 24 were tested for brain damage by a neuropsychiatrist who used the following methods: (1) case history, (2) neurological examination, (3) EEG and (4) photometrazol tests. Eight of the subjects were found to have cortical dysfunction, six were questionable, and in ten there was no evidence of cortical dysfunction. This study has the advantage of specifying the procedure used to determine brain damage but it is limited in that specific neurologic findings were not presented and the subclassifications were not

given. For example, in how many was there positive evidence from all four diagnostic procedures? Or, if positive evidence was obtained by one procedure, were the other procedures employed? It is not made clear whether the authors were using the terms "dysfunction" and "damage" interchangeably. The answers to questions such as these have been potential for changing rather nebulous classifications to well-documented neurological impairment which might lead to some understanding of brain-behavior relationships.

Miller (1964) reported on an attempt to teach an emotionally disturbed brain-injured child. There are limitations of using a single case. Generalizing from an n of 1 is dangerous, but that is not the main weakness of this study. In fact, much might be learned from a single patient where there is an accurate description of teaching procedures and good documentation of the brain injury which would include type and site of lesion, age of onset, premorbid history if possible, together with neurological information used in

making the diagnosis. In contrast, the patient was described as chronological age 8, having a normal appearance with a history of early illness with a high fever. (The degree and length of time the fever lasted were not specified.) The patient had an IQ of 80 (tests not specified) with the verbal IQ much lower than the performance IQ. The patient had a poor attention span and poor concentration. There appeared to be diffuse organic impairment. Behavioral symptoms included disorganized and impulsive behavior, drooling, and poor directional orientation. (The name of the test for directional orientation was not given.) The foregoing description was followed by the unusual statement that the EEG and neurological examination were inconclusive but indicated brain damage. No further details were given. The limitation of this study is that the behavioral symptoms are not synonymous with brain damage. The fact that the verbal IQ was lower than the performance IQ is irrelevant as far as providing diagnostic information about the brain. If the EEG and neurological were indeed inconclusive, it is questionable

whether they indicated brain damage. In other words, on the basis of the descriptive information listed for this case, the reader cannot determine whether brain damage was present. It is not only impossible to generalize to other children, it is almost impossible to know what the particular characteristics of this child were.

Silver and Hagin (1965, 1967 and 1967a) reported a long term experiment on the treatment of children with specific reading disabilities. There were forty boys divided into two groups with each boy paired in terms of chronological age, intelligence quotient, psychiatric diagnosis, and neurological status. The neurological status was determined from the classical neurological examination plus right-left discrimination, a measure of handedness, eyedness, and footedness and an extension test. These studies represent a well organized attempt to evaluate specific teaching procedures, i.e. stimulation of deficit perceptual areas, however too little information is given concerning the findings on the neurological examination. One is unable to determine the relation of neurological im-

pairment to improvement in reading, albeit this was not the author's purpose in doing the study.

Hewitt, Mayhew and Rabb (1967) attempted to develop a basic sight vocabulary in neurologically impaired, mentally retarded, and severely emotionally disturbed children. There were a total of 26 Ss, eight of whom were neurologically impaired. The neurological impairment was based on a history of convulsions, perceptual-motor difficulties and cerebral palsy. A technical point in this study which is frequently overlooked by non-neurological specialists is that convulsions or a seizure disorder alone is not necessarily diagnostic of brain damage, even in the presence of abnormal EEG tracings. It is true that many types of damage to the brain will result in seizures, focal or generalized, but the mere presence of seizures cannot be used to justify a diagnosis of brain damage. A seizure may result from an electrical disturbance where there is no evidence of damage. Indeed, as a consequence of a severe febrile condition, an otherwise normal patient may have a

convulsion. What needs to be specified in the study of brain damage is a condition which would result in the seizure.

Willson (1968) reported on the effectiveness of three clinical techniques applied to children for each of whom the most probable cause of reading retardation had been determined. She had three groups of male dyslexics consisting of 6, 5, and 6 Ss respectively, one group of which included the dyslexics with evidence of neurological disorders. There was no evidence as to how the neurological disorders were identified. There was only a statement.

Weiner (1969) reported on the effectiveness of resource rooms for children with specific learning disabilities. There were 72 Ss screened on the Wechsler Intelligence Scale for Children, the Bender-Gestalt, Draw-A-Person, Vineland, a neurological and a psychiatric examination. These Ss displayed classic symptoms of neurologic impairment: hyperactivity, dissociation, figure background reversals, distractibility, perseveration, and behavioral disorders. All were in the IQ range of 90-130, chronological age 7-12,

grade in school 1-6. No other information concerning the nature of the group was reported. In spite of the fact that a neurological examination was given, how the findings were used was not explained. The diagnosis of neurological impairment was apparently based on behavioral symptoms and as was stated previously, in classical neurology there is no known pathology which results in such a syndrome. A suspected limitation with respect to inferring dysfunction at the level of the cerebral hemispheres can be found in the reported IQ range of 90-130. It is very difficult to compose a group of brain-damaged children where the mean IQ will fall well into the average range for the general population. One of the resultants of damage, that is, actual tissue destruction at the level of the cerebral hemispheres is a general lowering of intellectual ability. (Reed and Reed, 1967, Reed and Fitzhugh, 1966, Reed, Reitan and Kløve, 1965). Weiner did not actually state he was studying brain-damaged children; rather, his interests were concerned with

children who had specific learning disabilities. Nevertheless, from his description of the sample, one could easily infer that he was interested in children with cortical involvement.

These seven studies represent experimental efforts that were made to investigate the relation of brain damage to reading. Another series of reports do not deal directly with children who are brain damaged but nevertheless, have implication for those interested in modifying instructional procedure as a consequence of neurological findings. Certain books have appeared describing procedures which may be used for teaching brain-injured children and these procedures represent either the wisdom of experts or the accumulated folklore of history, depending upon one's point of view.

Theories and Opinions

Delacato (1966) summarized his theory of neurological organization as it pertained to reading and presented a series of studies which reported on the efficacy of his training methods. Glass and Robins (1967) have reviewed these studies with respect to meeting

the criteria of sound experimentation. The training procedures may or may not be effective but in view of present knowledge, the development of the child from a neurological point of view does not provide justification for Delacato's methods. We do not know of any neurological evidence to support either (a) Delacato's stages of neurological organization or (b) use of his procedures as pedagogical devices for the improvement of reading.

Certain other writers (Ebersole, Kephart, Ebersole, 1968; Kephart, 1966; Orton and Gillingham, 1966) employ a vocabulary which smacks of neurological sophistication but which, in fact, might be quite misleading with respect to understanding how the condition of the brain may be related to reading or how teaching procedures may be differentially employed with brain-damaged youngsters. Orton, for example, theorized that certain reading disabilities were due to physiological variation related to the establishment of a normal unilateral dominance in the visual language area of the brain. There is no scientific evidence for such a statement. It

further implies that there is agreement that areas of the brain can be pinpointed and their functions specified. Such is not the case. Kephart (1966, pp. 171-180) emphasizing the concepts of space-time structures and the perceptual-motor match describes three major principles which must be followed in teaching brain-injured children. These principles include (1) developmental teaching from motor to perceptual to conceptual levels; (2) emphasis on the development of generalizations at all levels; and (3) establishment of veridicality in the already existing body of the child's information."...is desirable to use the strongest area of activity as the basis for establishing veridicality." (p. 177). These statements with their esoteric vocabulary imply more knowledge than actually exists. Like Spanish dubloons and pieces of eight, the scientific basis for such remarks is difficult to locate. Indeed, there were no studies where the concepts had even been put to a test. These and other writers do describe procedures which

for an individual child may prove to be effective. However, the effectiveness of the method may be quite separate from what the authors theorize as the justification for it. Above all, there is no empirical bases for recommending certain pedagogical procedures over other ones for use with brain-injured children as opposed to non-brain injured children who also may have a learning disability.

Cruickshank (1966) summarized and edited a book entitled The Teacher of Brain-Injured Children: A Discussion of the Base for Competency. The scope of the book went far beyond the teaching of reading alone yet the experimental evidence for many of the points of view expressed is almost nonexistent. The contributions of this volume may represent expert opinion and may represent effective programs of educational management. In general, though, they do not represent procedures grounded in sound experimental evidence. It is difficult to see, and perhaps unrealistic to try, how these methods and procedures represent unique programs for the brain-damaged child.

Summary and Recommendations

To change position from that of destructive critics to constructive critics, the following remarks are offered. Principals, curriculum supervisors, and teachers concerned with the education of brain-injured children, rather than going forward on the élan of acceptance and faith, should be quite critical of procedures which involve major reorganization of existing facilities, such as the restructuring of physical space, the dislocation of the individual child or the purchase of large, space-occupying training materials. Recommendations for teaching brain-injured children as defined within the context of this paper are only recommendations and little evidence exists for their support. If a child with a reading problem has suffered brain damage, and this damage has resulted in a neurological condition which is chronic and static in course, there is little evidence, if any, to suggest that the teaching procedures for such a child should differ materially from those used for another child with a reading problem of similar extent and degree but without

brain damage. Perhaps it is better to concentrate on developing aptitudes for reading rather than using procedures which essentially duplicate the teaching methods employed in the classroom and by which the child has already experienced failure. This is a question quite apart from the presence or absence of chronic cerebral dysfunction.

From the standpoint of research, it may well be that the classroom is not the appropriate laboratory or forum for experimentation, for clarification and eludication of brain-behavior relationships. If significant advances are to be made in teaching procedures for the brain-injured child, if there are to be effectively planned programs according to individual differences, and if there are to be precise evaluations of such programs, then during the next ten years more definitive experiments will have to be done than were performed during the decade of 1960 to 1970. An educational climate which produces so many recommendations and which

leads so many to engage in behaviors for which there is little experimental evidence is indeed worthy of sociological investigation. Good experiments on instructional procedures are extremely hard to design. Unravelling the effect of differential methods of instruction and relating these effects to the type and site of lesion may not in fact be possible because of the moral restrictions imposed in experiments with human beings as well as difficulties in studying any chronic disease of long duration. However, no progress can be made unless there is acute awareness of the necessity to provide adequate criterion information on the group studied.

One of the purposes stated at the start of this paper was to examine the effectiveness of methods used to teach reading to brain-injured children. This purpose was not achieved because of (a) the few number of experiments directed toward the problem, and (b) the inadequacy of the neurological information used to document brain damage. To determine whether children with organic cerebral neurological impairment, static in course, require or will benefit

from specially adapted teaching procedures, carefully designed experiments will have to be performed. Brain damage should be the independent variable and rate of progress or achievement level should be the dependent variable. Brain-damaged subjects will have to be studied along with non-brain-damaged subjects as controls.

The following recommendations are given:

1. The diagnosis of brain damage ought to be limited to those cases where there is strong reason to believe that tissue change has occurred. In any event, the detailed basis for group composition must be specified.

2. If the findings are based on the clinical neurological examination, the exact criteria specifying the abnormality should be stated. If more than one finding is present, the number and percent of the children showing each pathological sign should be specified.

3. When ancillary tests are used, the kind and type of test should be clearly stated, thus, if contrast studies were employed,

the number of children for whom this procedures was used and the details of the contrast studies should be given. If EEG was done, all details of the technique must be reported. If the information results from neurosurgical findings, the exact findings should be listed.

4. If groups heterogeneous with respect to brain damage are studied, the type of each neurological condition should be identified, and the number of children having this condition be specified. For example, in a group of ten children, four might have a documented history of head trauma, three might have suffered from an inflammatory disease, and three suffered from acute infantile hemiplegia. The age when the event occurred is necessary.

5. Anamnestic information is weak, but if it must be used, the specific criteria for acceptance or rejection should be delineated. It is not enough to say there was prolonged labor, the time interval should be given. Reporting only a history of high fever is useless. If hospital charts are used for information it should be

recognized that they are highly unreliable and a careful evaluation and reading of the chart must be done.

6. For the purpose of research, results from psychological tests cannot be accepted as evidence for brain damage in our present state of knowledge. The effects of brain lesions on psychological testing can vary with type and size of lesion, chronicity and acuteness, age of onset and a host of other variables too numerous to outline here. Furthermore, if a neurologist makes use of psychological test findings in the neurological examination, this fact should be specified. It is permissible to study the relation of teaching procedures to distortion of Bender-Gestalt drawings. To report distortions in Bender-Gestalt drawings as evidence for brain damage is naive and reveals a lack of appreciation for the complexities of obtaining neurological criterion information.

7. The selection of appropriate control Ss is an extremely complicated but an extremely crucial matter in a well designed study. There are many specialized diagnostic procedures which the physician

may not employ to rule out the presence of brain damage. Theoretically, it is desirable to use the same tests to rule out brain damage that are used to determine brain damage. In practice, this cannot be done. When control subjects are used, careful histories should be taken and perhaps the clinical neurological examination can be given, if there is no reason to suspect cerebral dysfunction either from the history or from the clinical examination. The Ss could be legitimately included as controls. However, it should be remembered that "absence of evidence is not evidence for absence".[2]

The foregoing recommendations are somewhat more strict than those advocated by the task force. Following them may be somewhat rewarding in the advancement of knowledge. To carry out the foregoing recommendations, the scientific investigator in reading will

[2] A comment made by Hans-Lukas Teuber, Ph.D. at a conference on The Late Effects of Head Injury, Washington, D.C., March, 1969.

require the active cooperation of one trained in the neurological sciences. The neurologist, the neurosurgeon, or the neuropathologist is the handmaiden who must provide the information and data concerning the condition of the brain and, of equal importance, give the limitations of such data. The rigorous criterion information provided by well executed contrast studies is no better than the man who interprets the roentgentogram. Knowledge of brain damage and reading will advance in proportion to the care exercised in specifying the criterion. A problem may well confront the investigator who does not have available the resources of a neurological laboratory, but who sincerely wishes to investigate or experiment on the effectiveness of teaching methods for brain-damaged children. There is one recommendation. Don't.

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